

University of Groningen

Mesenteric stenosis, collaterals, and compensatory blood flow

van Petersen, Andre S.; Kolkman, Jeroen J.; Meerwaldt, Robbert; Huisman, Ad B.; van der Palen, Job; Zeebregts, Clark J.; Geelkerken, Robert H.

Published in:
Journal of Vascular Surgery

DOI:
[10.1016/j.jvs.2014.01.063](https://doi.org/10.1016/j.jvs.2014.01.063)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2014

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

van Petersen, A. S., Kolkman, J. J., Meerwaldt, R., Huisman, A. B., van der Palen, J., Zeebregts, C. J., & Geelkerken, R. H. (2014). Mesenteric stenosis, collaterals, and compensatory blood flow. *Journal of Vascular Surgery*, 60(1), 111-119.e2. <https://doi.org/10.1016/j.jvs.2014.01.063>

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

Mesenteric stenosis, collaterals, and compensatory blood flow

André S. van Petersen, MD,^{a,f} Jeroen J. Kolkman, MD, PhD,^{b,h} Robbert Meerwaldt, MD, PhD,^a Ad B. Huisman, MD, PhD,^c Job van der Palen, MSc, PhD,^{d,e} Clark J. Zeebregts, MD, PhD,^g and Robert H. Geelkerken, MD, PhD,^a Enschede, Oss-Uden-Veghel, and Groningen, The Netherlands

Background: The mesenteric circulation has an extensive collateral network. Therefore, stenosis in one or more mesenteric arteries does not necessarily lead to symptoms. The objective of this study was to determine the effect of collateral flow on celiac artery (CA) and superior mesenteric artery (SMA) duplex parameters.

Methods: Between 1999 and 2007, a cohort of 228 patients analyzed for suspected chronic mesenteric syndrome was studied. Stenosis of the mesenteric vessels and collateral flow patterns were identified on angiography and categorized. The effect of stenosis in one mesenteric vessel and the presence of collaterals from the other unaffected vessel was examined in both the CA and SMA.

Results: Stenosis of the CA resulted in a significantly higher peak systolic velocity (PSV) and end-diastolic velocity in the normal SMA without stenosis. This was also found for the CA without stenosis in the presence of a stenosis of the SMA. An incremental effect of the severity of the CA stenosis was found with a mean SMA PSV of 158 cm/s when normal and 259 cm/s when occluded. The presence of collaterals had a clear effect on duplex parameters of the angiographically normal SMA. In the presence of collaterals and a 70% CA stenosis, the PSV in the normal SMA was significantly higher ($P = .025$).

Conclusions: This study shows that stenosis in either the CA or SMA increases flow velocities in the other unaffected mesenteric artery. This increase was correlated with the presence of collaterals. Collaterals and stenoses in one of the mesenteric arteries may lead to mimicking or overgrading of stenosis in the other mesenteric artery. (J Vasc Surg 2014;60:111-9.)

Although arterial stenosis of the mesenteric circulation is common, ischemic complaints develop in a minority of patients. This is illustrated by the low estimated incidence of symptomatic stenotic disease, or chronic mesenteric ischemia, of 2.8 per 100,000 persons per year,¹ despite a high prevalence of chronic mesenteric disease of 3% to 18% in the few available large series.²⁻⁵ The development of ischemic complaints does not depend on vessel stenosis alone. We have demonstrated the presence of ischemia, and successful treatment, in single-vessel involvement.^{6,7} In contrast, patients with two diseased vessels may remain asymptomatic for years, which may be explained by the

extensive collateral vascular pathways of the mesenteric circulation.⁸⁻¹² These mesenteric collaterals, embryonic remnants of vessels connecting the celiac artery (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA), can develop within one mesenteric artery outflow, between two mesenteric arteries, or between mesenteric and parietal or body wall vessels. The most common collateral pathways found between the CA and the SMA are the pancreaticoduodenal arcades and occasionally the arc of Buhler. Common connections between the SMA and the IMA include the marginal artery of Drummond and the more centrally located arc of Riolo.¹³ Although it is widely assumed that the abundant collateral network prevents development of symptomatic ischemic complaints in most subjects,^{8,12} the relation between stenosis and collateral flow changes has not been established.

In the carotid circulation, the role of compensational blood flow has been well demonstrated in unilateral occlusion.¹⁴⁻¹⁶ It was shown that the blood flow on the side of the internal carotid artery occlusion indeed depends on collateral vessels. The middle cerebral artery flow territory depended on increased flow in the vertebrobasilar arteries, and the anterior cerebral artery flow territories depended on the collateral flow by the unaffected internal carotid artery. Another effect of this increased collateral flow is overestimation of stenosis severity on the contralateral carotid artery if the standard flow velocity criteria are used.^{17,18}

In the mesenteric circulation, data on collateral flow patterns and compensational flow are limited. An animal study of Boley et al^{19,20} demonstrated that occlusion of

From the Departments of Surgery (Division of Vascular Surgery),^a Gastroenterology,^b Radiology (Division of Interventional Radiology),^c and Epidemiology,^d Medical Spectrum Twente, Enschede; the Department of Research Methodology, Measurement and Data Analysis, University of Twente, Enschede;^e the Department of Surgery (Division of Vascular Surgery), Bernhoven Hospital, Oss-Uden-Veghel;^f and the Departments of Surgery (Division of Vascular Surgery)^g and Gastroenterology,^h University Medical Center Groningen, University of Groningen, Groningen.

Author conflict of interest: none.

Additional material for this article may be found online at www.jvascsurg.org.

Reprint requests: André S. van Petersen, MD, Department of Surgery, Division of Vascular Surgery, Bernhoven Hospital, PO Box 707, 5400 AS Uden, The Netherlands (e-mail: a.vanpetersen@bernhoven.nl).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214/\$36.00

Copyright © 2014 by the Society for Vascular Surgery.

<http://dx.doi.org/10.1016/j.jvs.2014.01.063>

the SMA led to increased blood flow in both the CA and IMA. A small human study showed that SMA occlusion in five patients (three isolated cases of SMA occlusion and two with CA and SMA occlusions) was associated with an increase in IMA blood flow in all.²¹ From a large cohort of patients referred for potential symptomatic mesenteric stenoses, we investigated the effect of a stenosis in a single mesenteric artery on the blood patterns and collateral pathways by duplex ultrasonography and digital subtraction angiography. Mesenteric duplex and cutoff values have been described initially by Moneta²² and in patients with suspected chronic mesenteric ischemia by AbuRahma et al²³ and recently by our group.²⁴ We hypothesized that stenosis of one of the main mesenteric vessels, CA or SMA, might lead to increased compensatory blood flow in the unaffected vessel, probably related to collateral network development.

METHODS

In the period 1999 to 2007, 779 consecutive patients presented to our tertiary referral center for evaluation and possible treatment of chronic mesenteric ischemia. Patients were analyzed for chronic mesenteric ischemia as described before. All patients underwent standard workup including structured medical history, assessment of vessel anatomy, and function testing as previously described.²⁵ The final diagnosis was made by a multidisciplinary group, including a gastroenterologist and a vascular surgeon, as previously reported.^{7,24} For this study, patients with previous interventions of the mesenteric vasculature or aorta and nonelective patients were excluded, leaving 672 patients for analysis. Mesenteric artery duplex ultrasound imaging was performed in 531. In 324 of these, the clinical suspicion of chronic mesenteric ischemia was sustained and multiplane mesenteric angiography was performed. The duplex B-mode visualization was classified good (origin of CA and SMA clearly visualized without any doubt), moderate (origin of CA or SMA, or both, identifiable with minor doubt), or poor (origin of CA and SMA not identifiable). Only patients with good interpretable angiograms (with complete overview angiography and appropriate expiration and inspiration phases) and duplex scans were included in this study. The study did not meet the criteria for medical research with human subjects, as confirmed by the Institutional Review Board. Therefore, an informed consent procedure was not necessary.

Duplex ultrasound. Patients were scanned with a 3.5 MHz convex sector probe after a fasting episode of 6 hours to prevent postprandial flow changes.²⁶⁻²⁸ Duplex imaging was performed by registered experienced vascular technologists, supervised by a vascular surgeon or radiologist. Investigators were blinded to any angiographic results. The transabdominal duplex examination was performed in a standardized manner as described previously.²⁹ Doppler measurements were made with a sample size of 5 mm. Velocity measurements were taken with the smallest, most accurate flow-to-beam angle possible. No measurements were accepted if the angle was greater than 60°.

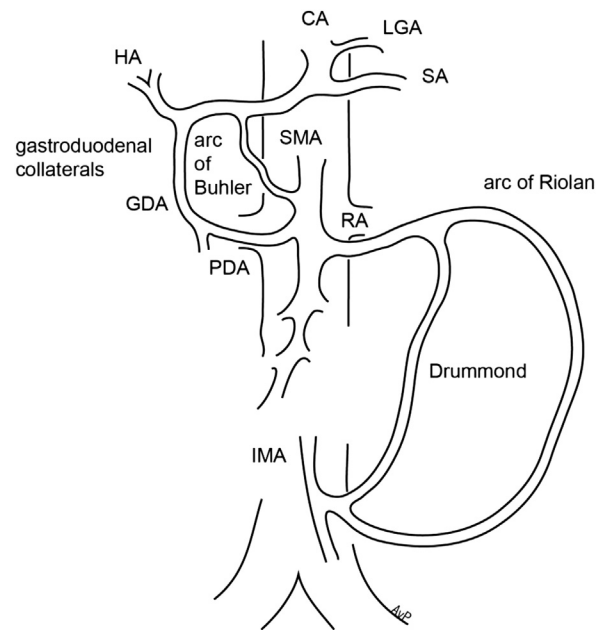


Fig 1. Studied collaterals between mesenteric arteries. CA, Celiac artery; GDA, gastroduodenal artery; HA, hepatic artery; IMA, inferior mesenteric artery; LGA, left gastric artery; PDA, pancreaticoduodenal artery; RA, renal artery; SA, splenic artery; SMA, superior mesenteric artery.

Doppler samples were taken during the maximum expiration and inspiration phases of respiration. Peak systolic velocity (PSV) and end-diastolic velocity (EDV) of the CA and SMA were determined. The findings were documented in written standard case record forms. Cutoff values for duplex imaging were used as described earlier²⁴: for PSV of the CA, 205 cm/s during expiration and 182 cm/s during inspiration; for PSV of the SMA, 220 cm/s during expiration and 169 cm/s during inspiration.

Abdominal angiography. Multiplane intra-arterial digital subtraction angiography of the abdominal aorta and its branches enabled multiple oblique projections of the abdominal aorta and of the origin and outflow of the mesenteric arteries. Angiography was performed during maximum expiration and inspiration phases of respiration. The manner in which analysis of angiographic findings was performed has been described previously.²⁴ The degree of stenosis and collateral vascularization on angiography during expiration and inspiration were determined retrospectively and independently by an interventional radiologist (A.B.H.) and two vascular surgeons (R.H.G., A.V.P.) who were blinded to the patient's symptoms and the results of the duplex ultrasound examination. The degree of stenosis was determined on both selective and nonselective angiography. In case of stenosis of the origin, nonselective angiography was more eligible to determine grade of stenosis. In all patients, collaterals were described for type and grade. A differentiation has to be made between normal arterial connections of the three mesenteric vessels and

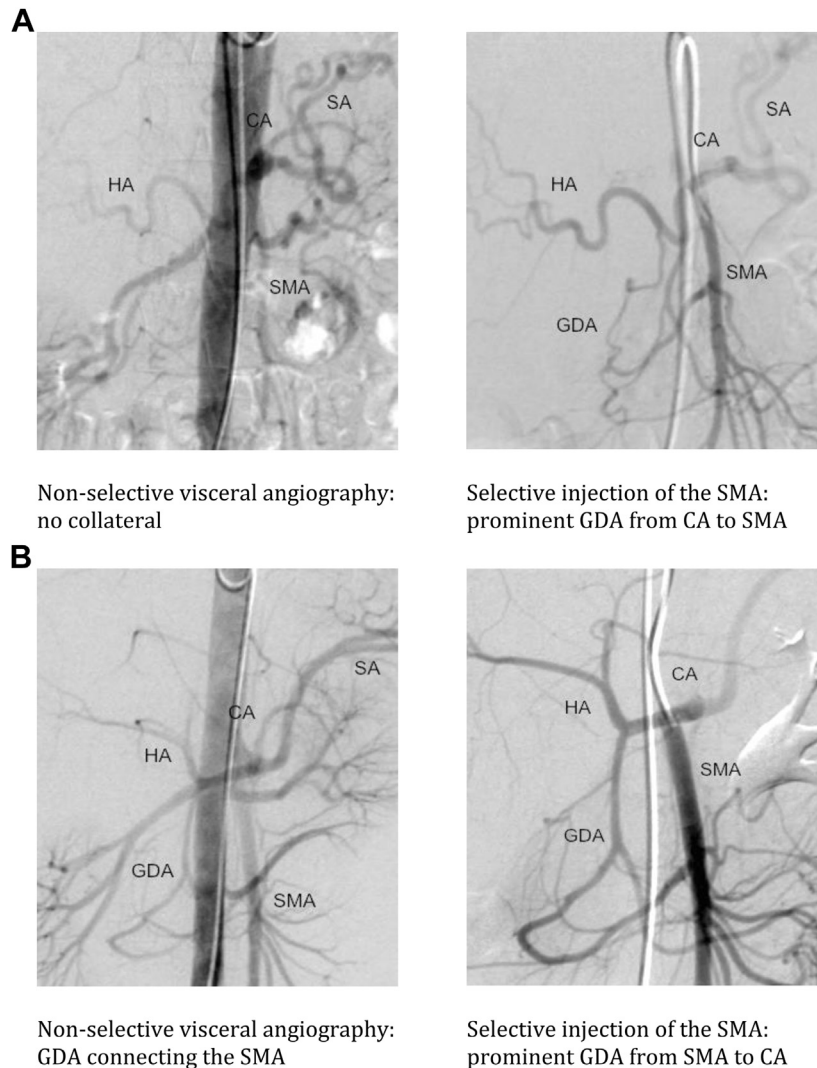


Fig 2. **A**, Collateral visible on selective injection only (grade 1) in a patient with stenosis of the celiac artery (CA). **B**, Collateral visible on nonselective visceral angiography (grade 2) in a patient with stenosis of the CA. GDA, Gastroduodenal artery; HA, hepatic artery; SA, splenic artery; SMA, superior mesenteric artery.

pathologic hypertrophic collaterals. We classified only interconnecting vessels as collateral when they were both clearly visible and hypertrophic. Collaterals found were categorized in one of the following groups: gastroduodenal, Buhler, Rioloan, and Drummond (Fig 1). For analysis of data, a new definition of collaterals is proposed. Collaterals were graded as visualized only during selective angiography (grade 1) or visualized prominently during nonselective visceral angiography (grade 2) (Fig 2).

Data management and statistical analysis. Interpretability of the duplex and angiography investigations was documented and categorized as good, moderate, and poor. In cases of occlusion and absent flow signal, data were excluded from analysis of the mean values. Comparison of angiograms and duplex ultrasound scans was performed with the same phase of respiration. To study

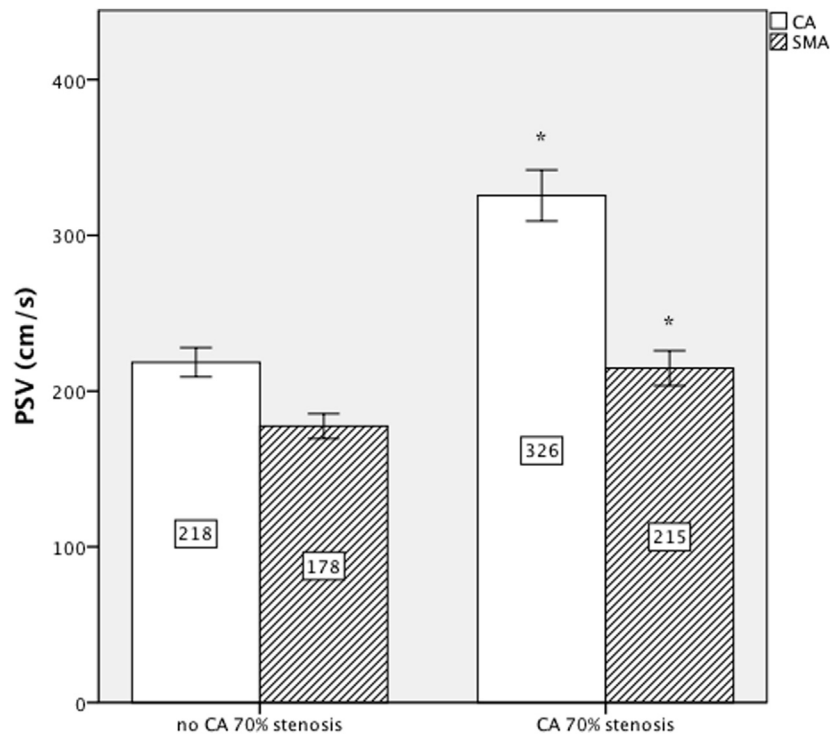
prevalence of collaterals, five different groups were analyzed: patients with normal findings on angiography, patients with <70% stenosis of the CA or SMA, patients with $\geq 70\%$ stenosis of the CA and $\leq 70\%$ stenosis of the SMA, patients with $\geq 70\%$ stenosis of the SMA and $\leq 70\%$ stenosis of the CA, and patients with $\geq 70\%$ stenosis of the CA and SMA. A subanalysis was performed in patients with an angiographically normal SMA and stenosis of the CA (normal, <50%, $\geq 50\%$ -70%, $\geq 70\%$ -99%, occlusion).

Data were expressed as mean and standard error of the mean. Independent-samples *t* test was used for comparison of duplex parameters between groups. Analysis of variance with post hoc Tukey test was used for comparison between multiple groups. All data were analyzed with SPSS 20 (SPSS, Chicago, Ill). Statistical significance was assumed for $P < .05$.

Table. Distinguished cohort of patients suspected to have chronic mesenteric syndrome with good interpretable duplex scans

	Vessel stenosis	Age (range), years	No. of patients	Collateral grade			Collateral type		
				% of N	Grade 1	Grade 2	GD	B	R/D
I	Normal	50 (18-81)	46	2	1	0			1
II	CA and SMA both <70%	51 (14-88)	65	20	11	2	7	1	10
III	CA ≥70% and SMA <70%	43 (15-79)	86	53	25	21	41	5	7
IV	SMA ≥70% and CA <70%	56 (22-72)	10	50	1	4	2		3
V	CA and SMA both >70%	61 (44-82)	21	86	3	15	3	1	15
Total		49 (14-88)	228	36	52	42	53	7	36

B, Buhler; CA, celiac artery; GD, gastroduodenal; R/D, arcade of Rioloan/Drummond; SMA, superior mesenteric artery. Data are shown as mean ± standard error of the mean.

**Fig 3.** Effect of stenosis in the celiac artery (CA) on duplex velocity parameters of the CA and superior mesenteric artery (SMA) during expiration. Data are shown as mean ± standard error of the mean. PSV, Peak systolic velocity.

* $P < .005$ comparing stenosis vs no stenosis.

RESULTS

The study cohort consisted of 228 patients suspected to have chronic mesenteric ischemia and with good interpretable duplex ultrasound scans and angiograms. Duplex data for expiration and inspiration were available in >90% of the patients with an ultrasound assessment of good interpretability. Normal findings on angiography of the CA and SMA were found in 46 patients (20%), stenosis <70% in both CA and SMA in 65 patients (29%), stenosis ≥70% in the CA and <70% in the SMA in 86 patients (38%), stenosis ≥70% in the SMA and <70% in the CA in 10 patients (4%), and stenosis ≥70% of both arteries in 21 patients (9%) (Table).

Group I: Normal inflow of CA and SMA. Completely normal findings on angiography of the CA and the SMA were found in 46 patients. A grade 1 collateral was observed in one patient with an IMA occlusion (Table). In this patient, duplex parameters of the CA and SMA were not different from those of patients with normal findings on angiography.

Group II: <70% stenosis in the CA or SMA. In patients with stenosis <70% in either the CA or SMA, collateral circulation was observed in 20%. Grade 1 collaterals were visualized in 85% of the cases (Table).

No significant changes were found in duplex parameters in the angiographically normal CA or SMA in

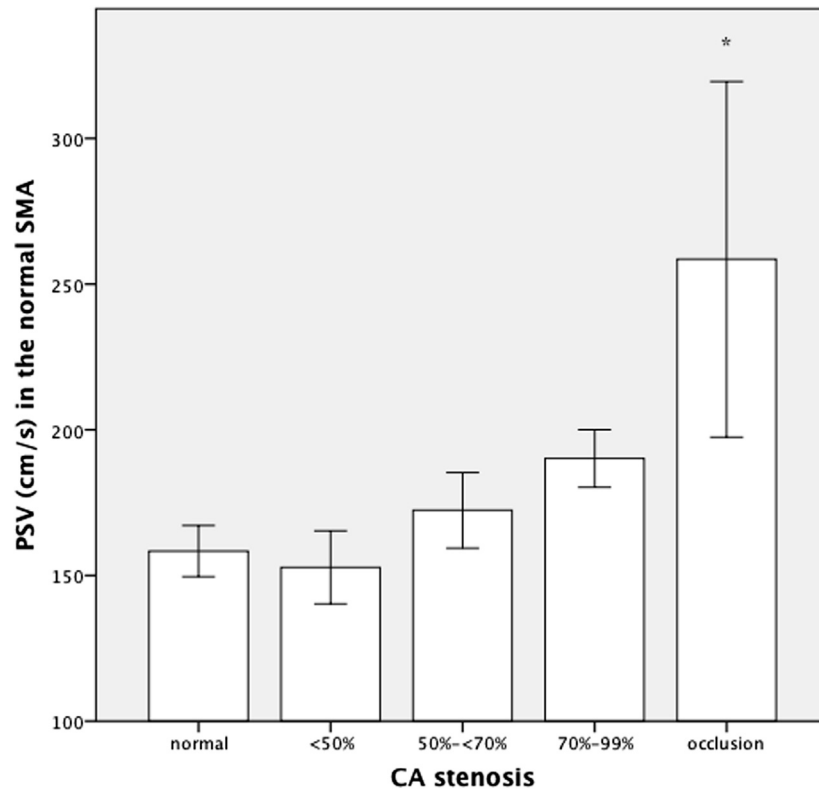


Fig 4. Patients with an angiographically normal superior mesenteric artery (SMA): effect of grade of celiac artery (CA) stenosis on SMA peak systolic velocity (PSV) during expiration. Data are shown as mean \pm standard error of the mean. $P = .009$, analysis of variance between groups; $*P < .05$, post hoc Tukey test compared with normal.

coexistence with a $<70\%$ stenosis in the other mesenteric artery (data not shown). Also, the presence of collaterals did not affect duplex parameters in this group.

Group III: $\geq 70\%$ stenosis in the CA. A $>70\%$ stenosis in the CA with an SMA with $<70\%$ stenosis was found in 86 patients. In 53% of these, one or more collaterals were present (Table). In 46% of these patients, this was a grade 2 collateral. The majority (89%) of the observed collaterals were through the gastroduodenal pathway.

The effect of a $\geq 70\%$ stenosis in the CA compared with patients with $<70\%$ stenosis or normal angiography findings on duplex parameters in the CA and SMA is shown in Fig 3 (Supplementary Table I, online only). The increase in PSV and EDV in the stenotic CA was as expected. However, in these patients, an increased PSV and EDV in the not significantly stenotic SMA was seen, during both expiration and inspiration (Fig 3 and Supplementary Table I, online only). To further study the effect of CA stenosis severity on the SMA blood flow, we performed a subanalysis of patients with an angiographically normal SMA. Of these 178 patients, during expiration 56 had a normal CA, 20 patients had a stenosis of $>50\%$ to 70% , 67 patients had a stenosis of $>70\%$, and 5 patients had an occluded CA. The effect of increasing stenosis of the CA on PSV

and EDV of the normal SMA is shown in Fig 4 (Supplementary Table II, online only). Patients with $\geq 70\%$ CA stenosis and a grade 2 collateral had a significantly higher PSV in the SMA compared with those without a collateral (Fig 5 and Supplementary Table III, online only); with grade 1 collaterals, only a trend toward higher PSV and EDV in the normal SMA was observed (Fig 6 and Supplementary Table IV, online only).

Group IV: $\geq 70\%$ stenosis in the SMA. A $\geq 70\%$ stenosis of only the SMA and not of the CA was found in 10 patients. In five patients, collaterals were seen on angiography, most of them (80%) grade 2.

With a $\geq 70\%$ SMA stenosis, the PSV and EDV increased both in the affected SMA, as expected, and in the unaffected CA (Fig 7 and Supplementary Table I, online only). The group of patients with an angiographically normal CA was small ($n = 55$), and just four patients had a concomitant stenosis of $\geq 70\%$ in the SMA. These numbers did not allow further subanalysis in patients with an angiographically normal CA as performed in group III.

Group V: $\geq 70\%$ stenosis in both the CA and SMA. Stenosis in both the CA and SMA was observed in 21 patients. A collateral circulation was observed in 86% of these

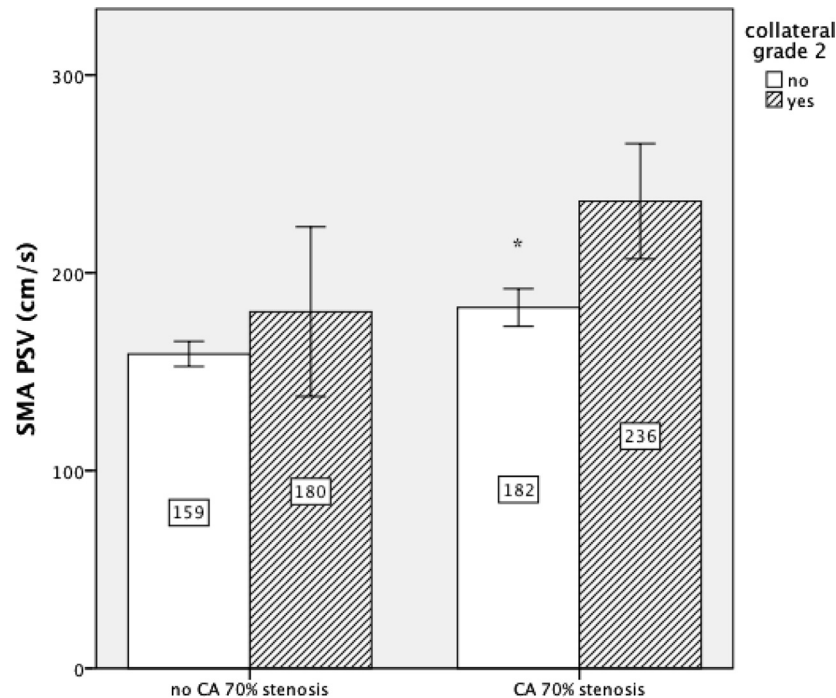


Fig 5. Patients with an angiographically normal superior mesenteric artery (SMA) with and without $\geq 70\%$ celiac artery (CA) stenosis: effect of existence of pathologic collateral pathways on SMA peak systolic velocity (PSV) during expiration. Data are shown as mean \pm standard error of the mean. * $P < .05$ comparing collateral vs no collateral and stenosis vs no stenosis.

patients; 83% of these were grade 2. In all of these patients, an arcade of Rioloan was visible. The number of patients was too low to reach statistical significance also because of the higher number of occlusions with consequently no duplex values.

DISCUSSION

This study is the first report of mesenteric arterial collateral pathways and their influence on CA and SMA duplex parameters in relation to mesenteric stenosis in a large cohort of 228 patients. The data from this study suggest that a stenosis grade $\geq 70\%$ in the mesenteric arteries may be the cutoff for collateral development and increased compensatory blood flow. Also, it emerges that not all collaterals are equal: collaterals observed during nonselective angiography always indicated important mesenteric stenosis, whereas grade 1 collaterals observed during selective injection were predominantly seen in normal and minimally stenosed vessels. In this study, a Rioloan artery was indicative of multivessel disease, whereas gastroduodenal collaterals were seen frequently in minimally stenotic vessels.

The distinction between collaterals shown on selective angiography (grade 1) or nonselective angiography (grade 2) and their clinical implications have, to our knowledge, not been reported before. The underlying physics involves the relative importance of the blood flow: with selective injection, the bolus may "push away" the normal blood flow, whereas collaterals viewed during nonselective

angiography indicate actual blood flow to the perfusion areas of the stenotic vessel. Indeed, grade 1 was observed predominantly in patients with less extensive mesenteric disease, whereas grade 2 was observed more in patients with severe stenosis in the CA and SMA. In patients with grade 2 collaterals, the flow in the unaffected SMA was higher compared with those with grade 1 collaterals, supporting the idea of compensatory blood flow. It seems plausible that grade 1 collaterals may develop to grade 2 with increasing stenosis from progressing mesenteric disease.

Collateral flow in patients with an isolated stenosis of the CA was facilitated mainly through the gastroduodenal pathway, in line with previous studies of patients with CA stenosis.¹² In the study of Libicher et al,³⁰ balloon occlusion of the CA was used as a pretest for endovascular covering. The most important collateral vessels observed in this study were the pancreaticoduodenal arcades and the dorsal pancreatic artery. The pancreaticoduodenal collaterals showed retrograde flow to the gastroduodenal artery and normal antegrade filling of the appropriate hepatic artery. The collaterals via the dorsal pancreatic artery were mainly responsible for filling of the splenic artery but showed also feeding vessels to the hepatic circulation. In patients with combined CA and SMA stenosis, collateral circulation was mainly facilitated through the arcade of Rioloan or Drummond. In this group of patients, flow in the IMA is likely to be higher. In our study, the IMA was not investigated by standard duplex examination. In

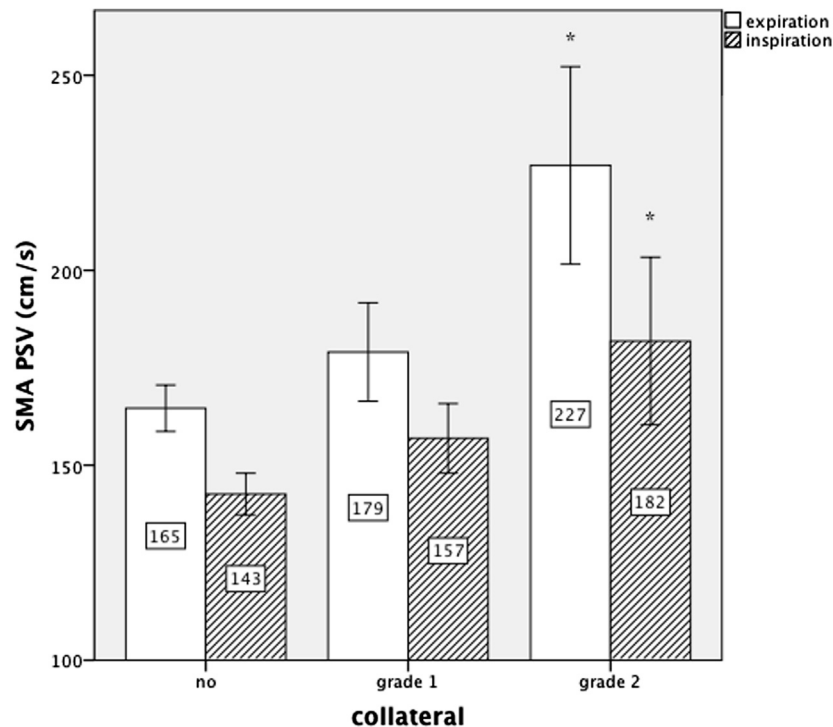


Fig 6. Patients with an angiographically normal superior mesenteric artery (SMA): effect of existence and type of collateral pathways on SMA duplex velocity parameters. Data are shown as mean \pm standard error of the mean. PSV, Peak systolic velocity. * $P < .05$ comparing groups (analysis of variance with post hoc Tukey test) compared with no collateral.

the presence of two-vessel disease, a significant rise in duplex parameters was found in the CA and SMA compared with comparable stenosis in single-vessel disease. This suggests that collateral flow from the IMA is not sufficient. As the grade of stenosis is comparable, a lower peripheral resistance in the mesenteric arterial circulation can only explain the increased PSV.

A differentiation of normal arterial connections between the three mesenteric vessels has to be made from pathologic hypertrophic collaterals. We have described collaterals on the basis of pattern of filling, when clearly visible and hypertrophic. It is likely that these collaterals have developed or are stimulated by the presence of a stenosis, stimulated by ischemia. We assume that they arise from small and physiologic embryonic remnants of interconnecting mesenteric arteries. However, we cannot exclude that these were already present before development of stenosis. The fact that these collaterals are not observed in this way in patients without stenosis, however, makes this unlikely. Most likely, these connections are available in most patients to be developed when needed.³¹ In the study of Kornblith et al,⁹ anatomic specimens of a broad selection of patients were correlated with angiography findings. An anatomic incidence of 2% was described for the arc of Buhler and with a visibility index of 2% infrequently visualized on angiography. Other collateral pathways were not found or described. The current understanding of collateral network

development involves a complex remodeling of preexisting conduit vessels and is probably driven by inflammatory processes.³² These inflammatory processes are triggered by ischemic events. Because an increase in collateral development was seen only in patients with $>70\%$ stenosis grade, this suggests that this is the minimum stenosis grade for development of ischemia. Whether the presence of collaterals is indicative of ischemia development, or conversely, that the lack of collaterals indicates the absence of ischemia needs to be confirmed.

The measured increased, compensational, blood flow is in line with the small studies available.^{20,21} Both studies found increased blood flow in the other mesenteric artery in the presence of SMA occlusion. Increased flow is most likely to be facilitated by collaterals and consequently decreased peripheral resistance. The effect of lower peripheral resistance on duplex parameters has been documented in the femoropopliteal bypass. It is known that vasodilating stimuli (eg, papaverine) are able to increase flow in bypass grafts.³³ In the study of Karacagil et al,³⁴ increase in PSV, mean velocity, and flow volume was found in autografts.³³ This effect has also been demonstrated in allograft bypasses, pointing to a decreased peripheral resistance causing an increased flow without change of diameter. In the study of Boley et al,³⁵ collateral flow was studied in dogs during experimental reduction of SMA flow. Flow rose in the CA and IMA after a reduction of flow in the SMA. This rise of

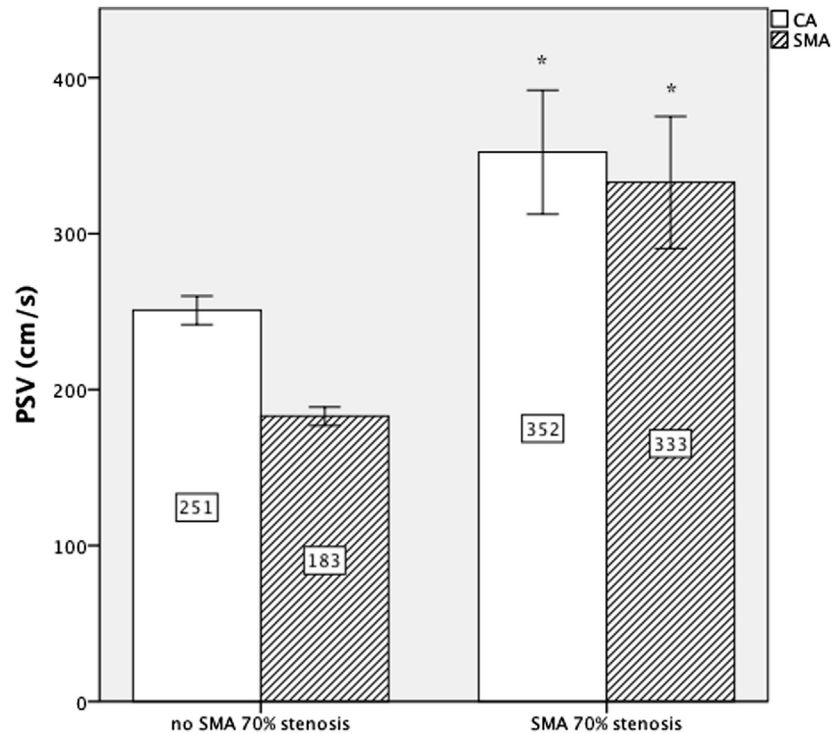


Fig 7. Effect of stenosis in the superior mesenteric artery (SMA) on duplex velocity parameters of the celiac artery (CA) and SMA during expiration. Data are shown as mean \pm standard error of the mean. PSV, Peak systolic velocity.

* $P < .005$ comparing stenosis vs no stenosis.

flow was more prominent in animals with intact collaterals, which evidently shows the function of collaterals. In a study in dogs after clamping of the SMA, Martin et al³⁶ found an increase in the rate of flow after the administration of papaverine. This effect of papaverine was found only in the presence of collateral vessels. Our findings are in line with these observations, although these studies are animal studies for acute mesenteric ischemia in contrast to our patient group with suspected chronic mesenteric ischemia.

One consequence of the increased blood flow parameters in the unaffected mesenteric vessel may be an overestimation of the stenosis severity in that vessel. A higher velocity was found in the nonstenosed mesenteric artery in patients with an isolated stenosis in both the CA and SMA. With use of the currently applicable cutoff values for duplex parameters to define mesenteric stenoses,²⁴ the flow velocities in the nonaffected vessels would have been classified as stenosis. If angiography had not been performed, this would have resulted in a false-positive diagnosis (stenosis $\geq 70\%$) in 40% of patients with a single SMA stenosis and in 7% of patients with a CA stenosis. Therefore, normal values should be viewed in the context of the other mesenteric vessel assessment. This also emphasizes the importance of B mode. An increased flow velocity that is not focal or associated with visualized stenosis should not necessarily be diagnosed as a stenosis. If flow velocities were used without B mode for diagnosis of a 70%

stenosis, the percentage of incorrect diagnoses would have been higher (respectively, 70% and 36%). This could also explain the differences in published cutoff values for mesenteric duplex parameters^{23,24} because a higher proportion of patients with one-vessel stenosis may lead to more patients with higher flow in nondiseased vessels within the cohort and thus higher normal cutoff values.

The limitation of this study is the retrospective analysis during a prolonged time. However, because data on duplex ultrasound and angiography studies were collected in a standardized, prospective fashion, this probably has little effect on the main findings. Another limitation is that we included only patients referred for suspected gastrointestinal ischemia; no healthy controls were studied. Finally, symptoms have not been analyzed for this study but are subject to further study.

CONCLUSIONS

This study shows that mesenteric stenosis above 70% leads to more collateral development and higher flow velocities in the unaffected vessel. Collateral vessels seen on nonselective angiography are indicative of significant mesenteric stenosis. When flow velocities are used as a measure of mesenteric stenosis, the effect of compensatory increased blood flow should be taken into account to avoid false-positive diagnoses.

AUTHOR CONTRIBUTIONS

Conception and design: AP, RG
Analysis and interpretation: AP, JK, JP, CZ, RG
Data collection: AP, JK, RM, AH, RG
Writing the article: AP, JK, CZ, RG
Critical revision of the article: AP, JK, RM, AH, JP, CZ, RG
Final approval of the article: AP, JK, RM, AH, JP, CZ, RG
Statistical analysis: AP, JP
Obtained funding: Not applicable
Overall responsibility: AP

REFERENCES

1. Kerkhof I, Kolkman JJ, Geelkerken RH, Brusse M. Incidence of chronic gastrointestinal ischemia: a large Dutch cohort study. *Gastroenterology* 2013;144:S902-3.
2. Roobottom CA, Dubbins PA. Significant disease of the celiac and superior mesenteric arteries in asymptomatic patients: predictive value of Doppler sonography. *AJR Am J Roentgenol* 1993;161:985-8.
3. Thomas JH, Blake K, Pierce GE, Hermreck AS, Seigel E. The clinical course of asymptomatic mesenteric arterial stenosis. *J Vasc Surg* 1998;27:840-4.
4. Hansen KJ, Wilson DB, Craven TE, Pearce JD, English WP, Edwards MS, et al. Mesenteric artery disease in the elderly. *J Vasc Surg* 2004;40:45-52.
5. Fried LP, Borhani NO, Enright P, Furberg CD, Gardin JM, Kronmal RA, et al. The Cardiovascular Health study: design and rationale. *Ann Epidemiol* 1991;1:263-76.
6. Mensink PB, van Petersen AS, Geelkerken RH, Otte JA, Huisman AB, Kolkman JJ. Clinical significance of splanchnic artery stenosis. *Br J Surg* 2006;93:1377-82.
7. Mensink PB, van Petersen AS, Kolkman JJ, Otte JA, Huisman AB, Geelkerken RH. Gastric exercise tonometry: the key investigation in patients with suspected celiac artery compression syndrome. *J Vasc Surg* 2006;44:277-81.
8. Fisher DF Jr, Fry WJ. Collateral mesenteric circulation. *Surg Gynecol Obstet* 1987;164:487-92.
9. Kornblith PL, Boley SJ, Whitehouse BS. Anatomy of the splanchnic circulation. *Surg Clin North Am* 1992;72:1-30.
10. Rosenblum JD, Boyle CM, Schwartz LB. The mesenteric circulation. Anatomy and physiology. *Surg Clin North Am* 1997;77:289-306.
11. Walker TG. Mesenteric vasculature and collateral pathways. *Semin Intervent Radiol* 2009;26:167-74.
12. Song SY, Chung JW, Kwon JW, Joh JH, Shin SJ, Kim HB, et al. Collateral pathways in patients with celiac axis stenosis: angiographic-spiral CT correlation. *Radiographics* 2002;22:881-93.
13. van Bockel JH, Geelkerken RH, Wasser MN. Chronic splanchnic ischaemia. *Best Pract Res Clin Gastroenterol* 2001;15:99-119.
14. van Everdingen KJ, Klijn CJ, Kappelle LJ, Mali WP, van der Grond J. MRA flow quantification in patients with a symptomatic internal carotid artery occlusion. The Dutch EC-IC Bypass Study Group. *Stroke* 1997;28:1595-600.
15. Fujitani RM, Mills JL, Wang LM, Taylor SM. The effect of unilateral internal carotid arterial occlusion upon contralateral duplex study: criteria for accurate interpretation. *J Vasc Surg* 1992;16:459-67; discussion: 467-8.
16. Forconi S, Johnston KW. Effect of contralateral internal carotid stenosis on the accuracy of continuous wave Doppler spectral analysis results. *J Cardiovasc Surg (Torino)* 1987;28:715-8.
17. AbuRahma AF, Richmond BK, Robinson PA, Khan S, Pollack JA, Alberts S. Effect of contralateral severe stenosis or carotid occlusion on duplex criteria of ipsilateral stenoses: comparative study of various duplex parameters. *J Vasc Surg* 1995;22:751-61; discussion: 761-2.
18. Kolkert JL, van den Dungen JJ, Loonstra J, Tiellu IF, Verhoeven EL, Beck AW, et al. Overestimation of contralateral internal carotid artery stenosis before ipsilateral surgical endarterectomy. *Eur J Radiol* 2011;77:68-72.
19. Boley SJ, Regan JA, Tunick PA, Everhard ME, Winslow PR, Veith FJ. Persistent vasoconstriction—a major factor in nonocclusive mesenteric ischemia. *Curr Top Surg Res* 1971;3:425-33.
20. Boley SJ, Brandt LJ, Veith FJ. Ischemic disorders of the intestines. *Curr Probl Surg* 1978;15:1-85.
21. Erden A, Yurdakul M, Cumhur T. Doppler waveforms of the normal and collateralized inferior mesenteric artery. *AJR Am J Roentgenol* 1998;171:619-27.
22. Moneta GL, Lee RW, Yeager RA, Taylor LM Jr, Porter JM. Mesenteric duplex scanning: a blinded prospective study. *J Vasc Surg* 1993;17:79-84; discussion: 85-6.
23. AbuRahma AF, Stone PA, Srivastava M, Dean LS, Keiffer T, Hass SM, et al. Mesenteric/cealic duplex ultrasound interpretation criteria revisited. *J Vasc Surg* 2012;55:428-36.e6.
24. van Petersen AS, Meerwaldt R, Kolkman JJ, Huisman AB, van der Palen J, van Bockel JH, et al. The influence of respiration on criteria for transabdominal duplex examination of the splanchnic arteries in patients with suspected chronic splanchnic ischemia. *J Vasc Surg* 2013;57:1603-11.
25. Otte JA, Geelkerken RH, Huisman AB, Kolkman JJ. What is the best diagnostic approach for chronic gastrointestinal ischemia? *Am J Gastroenterol* 2007;102:2005-10.
26. Volteas N, Labropoulos N, Leon M, Kalodiki E, Chan P, Nicolaides AN. Detection of superior mesenteric and coeliac artery stenosis with colour flow Duplex imaging. *Eur J Vasc Surg* 1993;7:616-20.
27. Gentile AT, Moneta GL, Lee RW, Masser PA, Taylor LM Jr, Porter JM. Usefulness of fasting and postprandial duplex ultrasound examinations for predicting high-grade superior mesenteric artery stenosis. *Am J Surg* 1995;169:476-9.
28. Mitchell EL, Moneta GL. Mesenteric duplex scanning. *Perspect Vasc Surg Endovasc Ther* 2006;18:175-83.
29. Delahunt TA, Geelkerken RH, Hermans J, Van Baalen JM, Vaughan AJ, Hajo Van Bockel J. Comparison of trans- and intra-abdominal duplex examinations of the splanchnic circulation. *Ultrasound Med Biol* 1996;22:165-71.
30. Libicher M, Reichert V, Aleksic M, Brunkwall J, Lackner KJ, Gawenda M. Balloon occlusion of the celiac artery: a test for evaluation of collateral circulation prior endovascular coverage. *Eur J Vasc Endovasc Surg* 2008;36:303-5.
31. Bertelli L, Lorenzini L, Bertelli E. The arterial vascularization of the large intestine. Anatomical and radiological study. *Surg Radiol Anat* 1996;18(Suppl 1):A1-6. S1-59.
32. la Sala A, Pontecorvo L, Agresta A, Rosano G, Stabile E. Regulation of collateral blood vessel development by the innate and adaptive immune system. *Trends Mol Med* 2012;18:494-501.
33. Karacagil S, Granbo A, Almgren B, Ljungman C, Bergqvist D. The effect of postocclusion reactive hyperaemia, papaverine and nifedipine on duplex derived haemodynamic parameters of infrainguinal bypass grafts. *Eur J Vasc Endovasc Surg* 1995;9:107-11.
34. Pedersen G, Laxdal E, Amundsen SR, Dregelid E, Jonung T, Nyheim T, et al. Flow measurement before and after papaverine injection in above-knee prosthetic femoropopliteal bypass. *J Vasc Surg* 2006;43:729-34.
35. Boley SJ, Treiber W, Winslow PR, Gliedman ML, Veith FJ. Circulatory response to acute reduction of superior mesenteric arterial blood flow. *Physiologist* 1969;12:180.
36. Martin WB, Laufman H, Tuell SW. Rationale of therapy in acute vascular occlusions based upon micrometric observations. *Ann Surg* 1949;129:476-93.

Submitted Nov 26, 2013; accepted Jan 25, 2014.

Additional material for this article may be found online at www.jvascsurg.org.

Supplementary Table I (online only). Effect of stenosis in either the celiac artery (CA) or superior mesenteric artery (SMA) on duplex velocity parameters (inspiratory and expiratory) of both vessels

	<i>Stenosis in CA</i>			<i>Stenosis in SMA</i>			<i>Normal</i>	<i>Stenosis ≥70%</i>
	<70%, (n = 121)	≥70%, (n = 107)	P ^a	<70%, (n = 197)	≥70%, (n = 31)	P ^a	CA and SMA, (n = 46)	CA and SMA, (n = 21)
PSV, cm/s								
CA, expiration	218 ± 9	326 ± 16	<.0005	251 ± 9	352 ± 40	.001	193 ± 16	417 ± 57
CA, inspiration	219 ± 9	334 ± 26	<.0005	232 ± 9	350 ± 45	<.0005	204 ± 25	451 ± 69
SMA, expiration	178 ± 8	215 ± 11	.006	183 ± 6	333 ± 42	<.0005	154 ± 9	350 ± 53
SMA, inspiration	157 ± 6	199 ± 17	.004	156 ± 5	296 ± 33	<.0005	135 ± 10	343 ± 50
EDV, cm/s								
CA, expiration	73 ± 5	124 ± 10	<.0005	88 ± 5	139 ± 29	.003	63 ± 8	176 ± 45
CA, inspiration	65 ± 4	135 ± 17	<.0005	72 ± 4	148 ± 32	<.0005	56 ± 8	209 ± 52
SMA, expiration	33 ± 2	55 ± 6	.0005	37 ± 2	117 ± 29	<.0005	29 ± 3	129 ± 41
SMA, inspiration	31 ± 2	53 ± 9	<.0005	31 ± 2	94 ± 17	<.0005	25 ± 2	115 ± 28

EDV, End-diastolic velocity; PSV, peak systolic velocity.

Values are mean ± standard error of the mean; duplex ultrasonography and angiography compared with same respiration phase.

^aComparing stenosis vs no stenosis (t test).**Supplementary Table II (online only).** Effect of stenosis in the celiac artery (CA) on duplex velocity parameters (inspiratory and expiratory) of the normal superior mesenteric artery (SMA)

	<i>CA stenosis</i>					P ^a
	<i>Normal</i>	<50%	50%-<70%	70%-99%	<i>Occlusion</i>	
Expiration	n = 56	n = 30	n = 20	n = 67	n = 5	
SMA PSV, cm/s	158 ± 9	153 ± 13	172 ± 13	190 ± 10	259 ± 61 ^b	.009
SMA EDV, cm/s	28 ± 2	29 ± 3	28 ± 2	43 ± 4 ^b	58 ± 15	.001
Inspiration	n = 66	n = 51	n = 25	n = 30	n = 5	
SMA PSV, cm/s	137 ± 8	153 ± 8	146 ± 11	159 ± 13	187 ± 32	.246
SMA EDV, cm/s	26 ± 2	30 ± 2	23 ± 2	35 ± 7	41 ± 6	.112

EDV, End-diastolic velocity; PSV, peak systolic velocity.

Values are mean ± standard error of the mean; duplex ultrasonography and angiography compared with same respiration phase.

^aComparing different groups (analysis of variance).^bP < .05 (post hoc Tukey test) compared with normal.**Supplementary Table III (online only).** Patients with an angiographically normal superior mesenteric artery (SMA) with and without ≥70% celiac artery (CA) stenosis: Effect of existence of pathologic collateral pathways on SMA peak systolic velocity (PSV) during expiration

<i>≥70% CA stenosis during expiration</i>	<i>Grade 2 collateral</i>		P	<i>Total</i>
	<i>No</i>	<i>Yes</i>		
No	159 ± 6 (96)	180 ± 43 (3)	.562	160 ± 6
Yes	182 ± 9 (54)	236 ± 29 (15)	.025	194 ± 10
P	.036	.428		.002
Total	167 ± 5	227 ± 25	.001	

Values are mean PSV (cm/s) ± standard error of the mean, and values in parentheses are number of patients.

Supplementary Table IV (online only). Patients with an angiographically normal superior mesenteric artery (SMA): Effect of existence and type of collateral pathways on SMA duplex velocity parameters

<i>SMA duplex parameters</i>	<i>Collateral</i>			<i>P^a</i>	<i>P^b</i>	<i>P^c</i>
	<i>No</i> <i>n = 128</i>	<i>Grade 1</i> <i>n = 31</i>	<i>Grade 2</i> <i>n = 19</i>			
PSV, expiration, cm/s	165 ± 6	179 ± 13	227 ± 25	.591	.068	.002
PSV, inspiration, cm/s	143 ± 5	157 ± 9	182 ± 21	.493	.366	.034
EDV, expiration, cm/s	31 ± 2	42 ± 7	50 ± 8	.080	.472	.005
EDV, inspiration, cm/s	27 ± 1	30 ± 2	43 ± 11	.631	.109	.006

EDV, End-diastolic velocity; *PSV*, peak systolic velocity.

Values are mean ± standard error of the mean. Analysis of variance with post hoc Tukey test.

^aBetween no collateral and grade 1.

^bBetween grade 1 and grade 2.

^cBetween no collateral and grade 2.